graft tissue, skin, to an abnormal culture atmosphere (45 % CO_2 in air) for various periods (7-48 h). Normally-cultured skin grafts caused no lymphoid malignancy. Immunogenicity of exposed grafts as a result of modification of cell-surface proteins and production of "altered self" structures may be a factor in the subsequent appearance of lymphoma in these autologous hosts.

In the animal models described above, malignant lymphoma develops as a late sequela of transplantations in the absence of exogenous immunosuppressants; chronic antigenic stimulation appears to be a possible common factor. Significantly, only lymphoid neoplasms developed despite the grafting of four different tissues—lymph node, mammary gland, skin, and bone marrow. Bone marrow is the only tissue that did not induce lymphoma in its recipients even following exposure to an unphysiological environment. This raises the question whether the T lymphocytes in the graft tissues are involved in the malignant processes in these animal models.

A E GOLDSMITH

National Cancer Cytology Center,
Melville, New York

G F RYAN

Brookhaven Memorial Hospital
Medical Center,
Patchogue, New York

A B JOSEPH

St Catharine's College,
University School of Clinical
Medicine, Cambridge

[1] Armstrong, M Y K, Schwartz, R S, and Beldotti, L, *Transplantation*, 1967, **5**, 1380.
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[3] Von Boehmer, H, and Byrd, W J, *Nature New Biology*, 1972, **235**, 50.
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Polymyalgia rheumatica and primary biliary cirrhosis

SIR,—May we comment on the paper by Dr J C Robertson and others (21 October, p 1128)? We have reported the careful rheumatological examination of 88 patients suffering from biopsy-proved primary biliary cirrhosis.[1] We found a much higher prevalence of rheumatic disorders than Sherlock and Scheuer[2] but did not find any patient who had the classical polymyalgia rheumatica syndrome. Polymyalgia rheumatica can be a difficult condition to diagnose, especially in patients who already have many other problems, but we took special care to elicit symptoms of stiffness and pain in the joints and muscles.

We did see three main rheumatological complications: scleroderma, avascular necrosis of the femoral or humeral head, and inflammatory arthritis. At that time we felt that the inflammatory arthritis was probably no greater than would be seen in a comparable population of middle-aged women. Further experience has suggested to us that we may not have been correct in this assessment and we hope to be reporting further on the inflammatory arthritis in primary biliary cirrhosis; but we doubt that the polymyalgic syndrome occurs sufficiently frequently in primary biliary cirrhosis to be an important association. We would also offer a word of caution about treating patients with corticosteroids with a doubtful diagnosis of polymyalgia as there is a real danger of accelerated osteoporosis.

However, we would make the point that patients presenting with a somewhat atypical rheumatological complaint should have, among other investigations, liver function tests performed with a view to the possibility that this might be an unusual manifestation of primary biliary cirrhosis.

A K CLARKE
ERIC HAMILTON
ROGER WILLIAMS

King's College Hospital,
London SE5 9RS

[1] Clarke, A K, *et al*, *Annals of Rheumatic Diseases*, 1978, **37**, 42.
[2] Sherlock, S, and Scheuer, M D, *New England Journal of Medicine*, 1973, **289**, 674.

Homoeopathic medicine

SIR,—Dr Hamish W Boyd's letter (24 March, p 821) is an extension of the old homoeopathic search for respectability.

Formation of a faculty, registration by Parliament, or registrable diplomas do not remove homoeopathy from fringe medicine. Those who feel that there may be a scientific basis for it should read recent publications on the subject such as those by Blackie[1] and by Ruthven Mitchell.[2] Both of these books speak a different language and indicate how little insight homoeopathic practitioners have into the powers of suggestion.

R S WALKER

Glasgow G3 7TD

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Postpartum haemorrhage and induction of labour

SIR,—We presented a paper (23 September 1978, p 855) showing an increased incidence of postpartum haemorrhage following induced labour compared with spontaneous labour. Additional data for the years 1975-7 from St Mary's Maternity Hospital, Portsmouth, confirm this finding.

A total of 10 320 spontaneous vaginal deliveries occurred in this three-year period, of which 6462 were spontaneous labours and 3858 were induced or accelerated labours. The accompanying table shows the figures for each year with the overall postpartum haemorrhage rate for the 10 320 cases. Multiparous and primiparous patients have been analysed separately, as they were in the initial study. The difference in postpartum haemorrhage rates between the induced and spontaneous groups is striking.

There has been no major change in the management of labour since our original paper was published. Intravenous prostaglandins have been used rarely for induction of labour. The oxytocin regimen remains unchanged and Syntometrine (ergometrine maleate and oxytocin) continues to be given for the third stage of labour.

MacKenzie (17 March, p 750) has confirmed our findings in his survey of delivery figures from Oxford. He has in addition been able to show the figures for accelerated labour as a separate category, which show a postpartum haemorrhage rate between that of spontaneous and induced labours.

We present these figures in support of our previous submission that postpartum haemorrhage should be considered as a serious complication of induction of labour, especially in primigravidae, and that it is probably related to the dosage of oxytocin infused.

P R BRINSDEN

Royal Naval Hospital,
Gosport, Hants

A D CLARK

St Mary's Maternity Hospital,
Portsmouth, Hants PO3 6AD

Postpartum haemorrhage (PPH) rates in induced and spontaneous labours: Portsmouth, 1975-7

	Spontaneous labour				Induced labour				
	Primiparae		Multiparae		Primiparae		Multiparae		
Year	Total No	No (%) with PPH	Total No	No (%) with PPH	Total No	No (%) with PPH	Total No	No (%) with PPH	Total
1975	731	27 (3·7)	1217	46 (3·8)	629	46 (7·3)	931	57 (6·1)	3508
1976	908	40 (4·4)	1451	62 (4·3)	448	37 (8·2)	702	49 (7·0)	3509
1977	838	33 (3·9)	1317	44 (3·3)	436	35 (8·0)	712	39 (5·5)	3303
Total	2477	100 (4·0)	3985	152 (3·8)	1513	118 (7·8)	2345	145 (6·2)	10320

Male sexual dysfunction and cimetidine

SIR,—I read with interest Dr N R Peden and colleagues' report (10 March, p 659) about three patients with sexual dysfunction after cimetidine and their postulation of an anti-androgenic action for cimetidine in man. It is worthy of comment that in two patients (cases 1 and 3) the luteinising hormone (LH) and follicle-stimulating hormone (FSH) levels were higher off cimetidine treatment and that the level of these hormones fluctuated at three and seven months after treatment in case 2. The data presented seem rather sparse to suggest overall trends.

Dr A M Hoare and I[1] were unable to find any differences in prolactin, total androgen, and oestradiol levels, or in the response of LH, FSH, and thyroid-stimulating hormone to appropriate releasing-hormone stimulation in 10 male subjects tested before and at completion of a 12-week course of cimetidine (1600 mg per day). Patients' ages ranged from 29 to 76 years (mean 50). Moreover, the lack of significant change is not altered by omitting those patients who were less than 40 years old.

It seems unlikely therefore that there is an endocrinological basis for the impotence reported by Peden *et al*, although other mechanisms may indeed be operating in the aetiology of this distressing complaint.

S G BARBER

The General Hospital,
Birmingham B4 6NH

[1] Barber, S G, and Hoare, A M, *Hormone and Metabolic Research*, 1979, **11**, in press.

Cimetidine and erythrosis-like lesions

SIR,—A 36-year-old man was admitted to our clinic on 2 November 1978 with a history of epigastric pain. Over the past 10 years he had had recurrent epigastric pain and three episodes of gastrointestinal bleeding after taking anti-inflammatory drugs.